Our Dermatology Online

Case Report

Vancomycin-induced linear IgA bullous dermatosis in a patient with chronic kidney disease: A case report

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ABSTRACT

Linear IgA dermatosis is a rare autoimmune blistering disorder characterized by linear IgA deposition along the basement membrane zone on direct immunofluorescence. In adults, it is most frequently drug-induced, particularly by vancomycin, and may clinically mimic severe cutaneous adverse reactions such as Stevens–Johnson syndrome, leading to diagnostic delays. We report a 41-year-old woman with end-stage renal disease on hemodialysis who developed vesiculobullous and targetoid lesions with mucosal involvement shortly after initiation of vancomycin for Staphylococcus epidermidis bacteremia. Direct immunofluorescence confirmed linear deposits of IgA, IgG, and C3 at the dermoepidermal junction. Prompt discontinuation of vancomycin resulted in complete resolution without recurrence. This case underscores the importance of early recognition of this entity in hospitalized patients, as timely diagnosis and drug withdrawal are essential to prevent unnecessary interventions and reduce morbidity.

Key words: Linear IgA Dermatosis, Vancomycin, Drug-Induced Skin Diseases, Bullous Dermatoses, Immunofluorescence, Direct

INTRODUCTION

Linear IgA dermatosis (LAD) is a rare autoimmune blistering disorder defined by linear IgA deposits at the dermoepidermal junction. In adults, it is frequently drug-induced, with vancomycin being the most common trigger.

Clinically, LAD can mimic severe reactions like Stevens-Johnson syndrome, often leading to diagnostic delays. Since the primary treatment is the immediate withdrawal of the offending agent, early recognition via direct immunofluorescence is essential. We report a case of vancomycin-induced LAD in a patient with end-stage renal disease to highlight the importance of prompt diagnosis in the hospital setting.

CASE REPORT

The patient was admitted to the internal medicine service for bacteremia due to coagulase-negative *Staphylococcus epidermidis* (identified via blood culture). Intravenous vancomycin therapy was initiated. Seventy two hours later, she developed erythematous vesicles and bullae with well-defined, regular borders, exhibiting targetoid and linear patterns across the face, trunk, and extremities. These findings were accompanied by bilateral axillary edema and painful oral mucosal erosions (Fig. 1a).

Initially, erythema multiforme secondary to a suspected herpes simplex virus infection was considered; however, the initiation of acyclovir yielded no clinical improvement. A skin biopsy was performed, and direct immunofluorescence (DIF) revealed concomitant linear deposits of IgA (++), IgG (++), and C3 (+) at the dermoepidermal junction (Fig. 1b), while IgM and C1q were negative. Based on the clinicopathological correlation, a diagnosis of vancomycin-induced linear IgA bullous dermatosis was established. Following the

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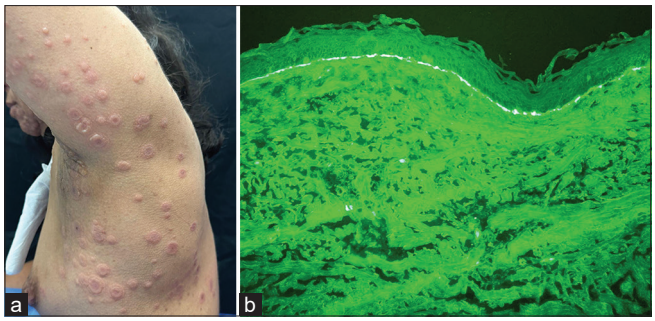

Figure 1: (a) Classic presentation of annular and target-shaped erythematous vesicles and blisters, called a "*string of pearls*" appearance. (b) Skin biopsy with direct immunofluorescence. Linear deposits of IgA (++), IgG (++) and C3 (+) at the dermoepidermal junction.

discontinuation of vancomycin and the initiation of supportive care, the cutaneous and mucosal lesions resolved within two weeks, with no recurrence post-discharge.

DISCUSSION

Linear immunoglobulin A (IgA) dermatosis, also known as linear IgA bullous disease (LAD), is a rare autoimmune subepidermal blistering disorder [1]. LAD is generally classified into two variants: a pediatric form, known as chronic bullous dermatosis of childhood, and an adult form, which can be idiopathic or drug-induced [2]. Immunopathologically, it is characterized by linear deposits of IgA along the dermoepidermal junction. The incidence ranges from 0.2 to 2.3 cases per million person-years, exhibiting a bimodal distribution with peaks in early childhood and after the sixth decade of life [1].

While most cases are idiopathic, drug-induced variants are well-documented, with vancomycin being the most frequently implicated agent [3,4]. Other associated medications include third-generation cephalosporins, trimethoprim-sulfamethoxazole, phenytoin, diclofenac, captopril, lithium, metronidazole, furosemide, atorvastatin, amiodarone, and amlodipine [5]. The latency period between drug exposure and symptom onset typically ranges from 1 to 21 days, often shortening upon re-exposure [1]. Notably, Gameiro et al. (2016) reported a latency of 3 to 15 days, with clinical improvement following drug withdrawal and resolution within 2 to 3 weeks without systemic therapy; however, the latter was observed in only one of the 44 reviewed cases [6].

Pathophysiologically, LAD involves autoantibodies directed against dermoepidermal adhesion antigens, such as BP180 (and its ectodomain LAD-1), BP230, type VII collagen, and laminin 332. In drug-induced forms, the exact mechanisms remain unclear; a hapten-mediated mechanism has been proposed, wherein the drug modifies basement membrane antigens to trigger an autoimmune response [3]. Several possible antigens have been detected by immunoblotting, including proteins of 97, 120, 180, 230, 250, and 285 kD [3]. Concurrent detection of IgG and C3 via immunofluorescence is a common finding and does not exclude the diagnosis [1,3].

Clinically, the disease presents with tense serous or hemorrhagic bullae, often arranged in an annular pattern or the classic herpetiform "string of pearls" configuration. It can also present as targetoid eruptions resembling erythema multiforme. Lesions commonly affect the extremities, palms, soles, trunk, and face; mucosal involvement occurs in up to 80% of cases, primarily affecting the oral and ocular mucosa [1,2,7]. Due to these morphological similarities, Stevens-Johnson syndrome is a critical differential diagnosis [1].

Direct immunofluorescence (DIF) remains the gold standard for diagnosis, demonstrating a sensitivity >90% and specificity of approximately 95% for linear IgA deposits. Recent reports suggest that in suspected LAD cases, deposits should be sought not only at the basement membrane but also around dermal vessels and eccrine glands [8]. While routine histopathology is a valuable adjunct, it lacks specificity and can mimic bullous pemphigoid. In diagnostically challenging cases, advanced techniques such as serration pattern analysis, salt-split skin indirect immunofluorescence, or antigen mapping may improve accuracy [3].

The mainstay of treatment for drug-induced LAD is the immediate discontinuation of the causative agent, which typically leads to resolution within 1 to 3 weeks [1]. In severe or refractory cases, dapsone is the first-line systemic treatment. However, it requires cautious administration in patients with end-stage renal disease due to the increased risk of methemoglobinemia and hemolytic anemia [2]. Alternative therapies include systemic corticosteroids, sulfonamides, colchicine, intravenous immunoglobulin (IVIg), mycophenolate mofetil, rituximab, and omalizumab [1,3].

The prognosis is generally favorable with early recognition and drug cessation. Conversely, delayed diagnosis can lead to unnecessary interventions and increased morbidity.

This case underscores the importance of including LAD in the differential diagnosis of hospitalized patients treated with vancomycin, particularly those on multiple medications who develop vesiculo-bullous eruptions. Direct immunofluorescence can confirm the diagnosis, allowing for immediate discontinuation of the drug, which is crucial for a good prognosis.

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